is the published standard of care in 1991 (1–4). While surgical palliation is not immune to complications, we believe that most major centers achieve similar results to those published recently by Kirklin et al. (5): 53 consecutive patients with tetralogy of Fallot and pulmonary stenosis who received an aortopulmonary shunt as palliation with a mortality of 0%. In the setting of a low surgical risk, coupled with the proved benefits of providing a shunt to a symptomatic child with tetralogy of Fallot (6), we cannot justify another palliative approach that does not protect pulmonary blood flow.

Finally, Guntheroth implies that decreased systemic vascular resistance and hyperventilation are at the root of hypercyanotic spells, and that the infundibulum is not necessary for their occurrence. He cites as evidence "spells" in patients with pulmonary atresia. In those patients, as in any patient with shunt-dependent pulmonary blood flow, a drop in systemic resistance results in a decreased "driving force" for systemic blood to enter the pulmonary circuit. In these patients, systemic desaturation associated with systemic vasodilation is often seen with high fever or when the child is placed in a hot bath. In tetralogy of Fallot, however, the frequency of spells induced by a catheter traversing the infundibulum in the laboratory, the effectiveness of beta-blocker therapy in the acute management of a spell, and the disappearance of the outflow tract murmur during a hypercyanotic spell cannot be explained solely by a drop in systemic vascular resistance (2). We concede that the mechanisms of hypercyanotic spells in tetralogy of Fallot are not fully understood, but we cannot accept Guntheroth's supposition that the infundibulum plays no role.

When Guntheroth states in his letter that there is no assurance of abolishing spells with medical or surgical palliation, is he implying that there is no protection afforded these patients from an aortopulmonary shunt? Certainly experience argues otherwise (6).

We maintain our strong opposition to balloon valvuloplasty as an alternative to an aortopulmonary shunt in symptomatic children with tetralogy of Fallot. In our opinion, it cannot ensure the adequacy of pulmonary blood flow, and potentially adds significant risk to an already difficult catheterization, even in the most experienced hands.

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Reply: II

I fully endorse the views of Guntheroth. First, I do not subscribe to the belief that cyanotic spells are due to "infundibular spasm"; rather, they are the result of a drop in the systemic vascular resistance. In our catheter laboratory, we now use phenylephrine, a potent alpha-adrenergic agonist, in preference to propranolol for relief of cyanotic spells during catheterization. Second, while a moderate degree of hypoxia is undesirable in the long term, we have endeavored to provide all patients with Fallot's tetralogy with corrective surgery in the 2nd year of life. Balloon pulmonary valvuloplasty appears to provide adequate palliation for many patients until this time, so that an aortopulmonary shunt may be avoided in the interim. This is a reasonable option, particularly as neonatal correction still carries a high mortality rate (1). Even with correction in the 1st year of life, a transannular patch is required in most cases. In the largest series with long-term follow-up, the use of a transannular patch was associated with persistent cardiomegaly in a significant proportion of patients, presumably because of significant pulmonary regurgitation (2). In our experience, delaying surgery beyond 1 year of age appears to decrease the requirement for a transannular patch. For these reasons, we continue to perform balloon pulmonary valvuloplasty as the initial palliative procedure in symptomatic infants with Fallot's tetralogy.

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Hemorrhaging Heart Patients

I realize in this age of randomized blind studies there is little place for the prejudices of a quarter of a century of taking care of patients with gastrointestinal bleeding, but I have the distinct impression that I see patients whose major hemorrhages are contact related in that tablets hold up in pyloric channels, duodenal bulbar and post-bulbar areas that have been narrowed by previous ulcer disease. This week I saw a man who had undergone two coronary angioplasty procedures, was taking two coated aspirins a day and had an unappreciated pyloric stenosis from old ulcer disease. His hemorrhage could not be controlled and required an antrectomy with a Billroth I for a 7 unit bleed. It will take a very large study to prove my thesis, but in the meantime upon arising I chew and wash away my some sort of buffered aspirin every other day on an empty stomach (I am in the National Physicians Study) and have a feeling your patients might do well to do the same. Hemorrhaging heart patients aren't fun!

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